A Dissertation on

GASTROINTESTINAL TRACT PERFORATIONS – TRAUMATIC & NON-TRAUMATIC

Submitted to
THE TAMILNADU DR. M. G. R. MEDICAL UNIVERSITY
CHENNAI

with fulfillment of the regulations
for the award of
M.S DEGREE IN GENERAL SURGERY
BRANCH I

GOVERNMENT RAJAJI HOSPITAL,
MADURAI MEDICAL COLLEGE, MADURAI

MARCH 2007

CERTIFICATE

This is to certify that the dissertation entitled **"GASTROINTESTINAL TRACT PERFORATIONS – TRAUMATIC & NON-TRAUMATIC",** is a bonafide work done by **Dr. R.SWAMINATHAN**, in **M.S BRANCH I GENERAL SURGERY** at Government Rajaji Hospital, Madurai Medical College, Madurai, to be submitted to The Tamil Nadu Dr.M.G.R Medical University, in partial fulfillment of the University Rules and Regulation for the award of M.S Degree Branch I General Surgery, under my supervision and guidance, during the academic period from July 2004 to July 2006.

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DECLARATION

I solemnly declare that this dissertation **"GASTRO INTESTINAL TRACT PERFORATION – TRAUMATIC & NON-TRAUMATIC"** was prepared by me at Government Rajaji Hospital, Madurai Medical College, Madurai under the guidance and supervision of **Prof.Dr.M.Kalyanasundaram, M.S., FICS,** Head of the Department of Surgery, Govt. Rajaji Hospital, Madurai Medical College, Madurai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the university regulations for the award of the degree of M.S. Branch 1 General Surgery.

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ACKNOWLEDGEMENT

I express my sincere thanks to the Dean **Dr. S.M.SIVAKUMAR, M.S.,** for permitting me to conduct the study in Government Rajaji Hospital, Madurai Medical College, Madurai

With deep sense of gratitude and respect, I take this opportunity to thank my teacher and professor **Dr. S.VIJIYALAKSHMI** M.S., for her constant inspiration, able guidance and kind help which she rendered in preparing this dissertation and in pursuit of my post graduate studies.

I am deeply grateful to **Prof. Dr.M.Kalyanasundaram**. M.S., FICS., Head of Department of Surgery, Government Rajaji Hospital, Madurai Medical College, Madurai for his constant encouragement and valuable guidance.

I am very grateful to all the senior civil surgeons, civil surgeons and Asst. surgeons of the Department of General Surgery, Government Rajaji Hospital, Madurai Medical College for their valuable guidance in conducting this study.

Last but not the least my heartfelt gratitude to the patients for their kind understanding and cooperation.

CONTENTS

INTRODUCTION

Gastro Intestinal Tract Perforations represent one of the most common acute abdominal emergencies in the surgical field. Differences in the clinical presentation of Gastro Intestinal tract perforations vary from the typical severe acute abdominal pain at one end, to subtle or no symptoms in the hospitalized patients for unrelated illness at the other end[1]. The various atypical presentations that mimic other abdominal conditions throw a real challenge over the diagnosis to the emergency surgeon.

A careful medical history, methodical clinical examination and radiological study plays a major role in the early diagnosis of this acute abdominal emergency. There are multiple factors that influence the prognosis and outcome of the patient. Preoperative resuscitation, intravenous administration of broad-spectrum antibiotics and good postoperative care are the mainstay in the management of Gastro Intestinal Perforations. The operative management depends upon the cause of perforations.

The present study deals with the etiology, clinical features, treatment modalities and factors influencing the prognosis of Gastro intestinal perforations at Government Rajaji Hospital, Madurai Medical College, Madurai.

AIMS OF THE STUDY

The aims of this study include:

1. To study the incidence, age and sex distribution of gastro intestinal perforations.
2. To study the etiology and clinical features of gastro intestinal perforations.
3. To study the different surgical techniques in the management.
4. To study the factors influencing the outcome of the patients.
5. To study the mortality and morbidity of gastro intestinal perforations.

REVIEW OF LITERATURE

Perforation of a hollow viscus is an acute abdominal emergency. Following perforation there is spillage of Gastro intestinal contents into the peritoneal cavity resulting in peritonitis, fluid and electrolyte imbalance, hypovolumia, circulatory insufficiency, septicemia and finally death.

A brief anatomy of the peritoneal cavity is described to understand the spread of liquid in the peritoneal cavity.

SURGICAL ANATOMY

The peritoneal cavity is lined with a single layer of mesothelial cells. The parietal peritoneum covers the abdominal cavity (i.e., abdominal wall, diaphragm, pelvis), the visceral peritoneum covers all the intra abdominal viscerae, forming a cavity that is completely enclosed except at the open ends of the fallopian tubes.[2]

A small amount of fluid sufficient to allow movements of organs is usually present in the peritoneal cavity. The fluid is normally serous (protein content <30 g/l, < 300 WBCs/μl). In the presence of infection, the amount of this fluid increases, the protein contents climbs to more than 30 g/l and the WBC count increases to more than 500 WBCS/μl, in other words, the fluid becomes an exudate.

The transverse colon and the drape of greater omentum divide the abdomen horizontally into supracolic and infracolic compartments. Therefore the symptoms and signs of peritonitis may be localized to upper or lower halves of the abdomen for sometime.

The forward convexity of the lumbar spine provides two marked lateral gutters and only a shallow anterior communication between them across the midline. Consequently, liquid spreads by movement largely around the periphery of the abdomen and not a great deal across the midline, hence the initial laterality of many peritoneal processes.

The right subhepatic space (Morison's pouch) is open only to the right, where it communicates with the right paracolic gutter. Liquid from perforated duodenal ulcer or seepage from the gallbladder region passes to the right and then both upwards to reach the right subphrenic space and downwards to the right iliac fossa. Thus, on one hand, there is subphrenic abscess and shoulder tip pain and on the other hand, the occasional diagnostic confusion between appendicitis and either perforated peptic-ulcer or acute biliary tract conditions.

Paracolic effusions reach the general peritoneal cavity across the sigmoid flexure. Pelvic effusions pass up both the paracolic gutters and there after to the subphrenic spaces and to the general peritoneal cavity.

A left sided origin above the transverse colon results in left paracolic and left subphrenic spread.[3]

AETIOLOGY

- Peptic ulcer disease complication.
- Acute appendicitis.
- Ingestion of nonsteroidal anti-inflammatory drugs (NSAIDs) usually observed in elderly patients.
- Infections:
    1. Typhoid fever complicated by intestinal perforation in about 5% of patients.
    2. Tuberculosis
- Inflammatory bowel disease
    1. Perforation in patients with ulcerative colitis.
    2. Perforation of terminal ileum in patients with Crohn's disease.
- Diverticular disease
- Meckel's diverticulum
- Perforation secondary to intestinal ischemia.
- Perforation occurring in patients with intra abdominal malignancy.
- Perforation due to injuries.
    1. Penetrating injury of lower chest and abdomen.
    2. Blunt injuries as in road traffic accident, handle bar injuries.
- Iatrogenic perforation of gastro intestinal tract.
    1. Endoscopy, ERCP, Colonoscopy
    2. Intestinal perforation as a complication of laparoscopy.
- Other rare causes
    1. Caustic ingestion

2. Foreign bodies
3. Blast wave injury and perforations
4. Rectal impalement injury (Bull gore injury)
5. Gunshot injuries

The gastro intestinal perforations are described under the following headings according to the anatomical locations.

1. Gastro duodenal perforations.
2. Perforations of the small bowel including the appendix.
3. Perforations of the large bowel.

GASTRO DUODENAL PERFORATIONS

AETIOLOGY

- Complications of peptic ulcer disease.
- Drug induced perforation
- Traumatic perforation
- Iatrogenic perforation
- Cushing ulcer perforation
- Curling's ulcer perforation
- Zollinger Ellison syndrome
- Malignant perforation: 10% of the perforations in the stomach are malignant. [4]

PEPTIC ULCER DISEASE

Background

Peptic ulcer disease of the stomach and duodenum has undergone dramatic evolution of over the past 40 years. Overall morbidity, hospitalization and operations for peptic ulcer disease has decreased[5], thanks to the widespread use of gastric antisecretory agents and H.pylori eradiation.

There has been a relative increase in the incidence of peptic ulcer disease in the elderly, resulting in increased morbidity and hospitalization in that age group, the elderly female has been the most profoundly affected largely because of use of NSAIDs in this population.[5]

The changes in the Peptic ulcer diseases have not been confined to the west. Report from India and elsewhere support the global trend towards decreasing incidence of peptic ulcer disease.

But trends in complication of peptic ulcer disease however have not shown the same decline[6]. There has been no parallel decrease in cases of duodenal ulcer with complications (Perforation, Hemorrhage, obstruction) and hospitalization for complications for gastric ulcer are increasing,.

ASSOCIATION OF HELICOBACTER PYLORI AND PEPTIC ULCER

It is now found that H.pylori is present in 90% of patients with duodenal ulcer and 75% of patients with gastric ulcer.[7] Infection appears to be acquired in the childhood and is inversely associated with socio-economic status.

H. pylori is a microaerophilic, spiral or helical Gram negative rod with 4 to 6 flagella. It resides in the gastric type epithelium within or beneath the mucus layer. It is one of the most potent producers of urease enzyme that is capable of splitting urea into ammonia and bicarbonate.

H.pylori induced gastrointestinal injury remains to be fully elucidated. Three potential mechanisms have been proposed.

i) Production of toxic products to cause local tissue injury.

ii) Induction of a local mucosal immune response.

iii) Increased gastrin level with a resultant increase in acid secretion.

The mucosal barrier is disturbed by the local immune response and the generation of large amount of ammonia leads to alteration in pH, mucosal charge gradient, cellular permeability, and epithelial sodium potassium ATPase activity leading to increased hydrogen ions.[8]

Diagnostic tests for H. pylori

- Non-invasive tests include serology and carbon labeled urea breath test.
- Invasive tests include rapid urease test, histology and culture but require the employment of endoscopy.
- Special stains such as Giemsa and Warthin starry silver stain are used for improved visibility than routine eosin and hematoxylin.

H.pylori infections show a strong association with peptic ulcer perforation. Eradication therapy directed against H. pylori promotes ulcer healing and prevents recurrence.

PEPTIC ULCER PERFORATION

Incidence

- The incidence of perforation of peptic ulcer is 7 to 10 cases per 1,00,000 population per year.[9]
- 7% of the patients hospitalized for peptic ulcer disease present with perforation.[9]

- Perforation is the first manifestations in about 2% of the patients with peptic ulcer diseases.
- Pyloroduodenal perforation occurs 6 to 8 times more often than gastric perforation.

Age

- Peptic ulcer perforations occur more commonly in the middle aged between 30 to 50 years.
- Now increasing use of NSAIDs have resulted in a shift in the incidence of perforation in the 6^{th} and 7^{th} decade of life.

Sex

- The sex distribution of peptic ulcer perforation shows a male : female incidence of 2:1.
- At present there is a steady increase in the number of females of the older age group using NSAIDs.
- Prepyloric perforations occur more often in young men where as gastric perforation is more common in the elderly women.[10]

Occupation

- Peptic ulcer perforations are more common in patients of low socio economic status.

RISK FACTORS

1. Use of NSAID
2. Smoking
3. Increasing patients age

4. Patients on immuno suppressive therapy
5. Chronic obstructive pulmonary disease
6. Major burns
7. Multi organ system failure.

PATHOPHYSIOLOGY

A peptic ulcer is said to have perforated when it extends through the muscle wall and serosa of the gastro intestinal tract thereby establishing communication between the lumen and adjacent space or structure. The perforation occurs as a result of sudden sloughing of the base of the ulcer due to impaired blood supply.

The site of pyloroduodenal perforations is usually the anterior wall and majority of the perforated gastric ulcers are located on the lesser curvature.[11] Posterior perforation of a gastric ulcer may occur into the lesser sac.

Perforation leads to leakage of gastric or duodenal contents into the peritoneal cavity initiating an acute peritonitis. Although it is an initial chemical peritonitis, bacterial peritonitis supervenes over the next few hours.

The presence of bacteria in the peritoneal cavity stimulates an inflow of acute inflammatory cells. The omentum and the viscera tend to localize the site of inflammation. This results in an area of localized hypoxia, which in turn facilitates growth of anaerobes and produce impairment of bactericidal activity of granulocytes. This leads to increased phagocytic activity of granulocyte, degradation of cells, hyper secretion of fluid forming the abscess, osmotic effects, shift of more fluids into the abscess area and enlargement of the peritoneal exudates causing paralytic ileus.

Absorption of bacterial endotoxins through the inflamed peritoneal surface causes endotoxemia. The combination of fluid and electrolyte imbalance and septicemia results in shock and multi organ failure, which is the cause of, increased mortality in untreated patients of perforative peritonitis.

MICROBIOLOGY

The microbiology of the Gastro intestinal tract changes from its proximal to its distal part. Few bacteria populate the proximal part of the bowel, where as the distal bowel contains aerobic organisms and higher percentage of anaerobic organisms. The common organisms are Escherichia coli and Bacteroides fragilis.

CLINICAL FEATURES

Peptic ulcer perforation are classified into

- Acute free perforation
- Confined perforation / chronic penetration.

ACUTE FREE PERFORATION

The clinical course is classically divided into 3 stages.[12]

- Primary stage - Stage of peritoneal irritation.
- Secondary stage - Stage of peritoneal reaction.
- Tertiary stage – Stage of bacterial peritonitis.

STAGE OF PERITONEAL IRRITATION

Also known as peritonism. This stage lasts for the first 2 to 3 hours following perforation. The sudden outpouring of caustic gastric juice into the peritoneal cavity producing chemical peritonitis causes the initial symptoms. The patient can recall the exact time of perforation by the abrupt on set of intense abdominal pain. The patient may or may not vomit. Referred pain is felt over the

tip of left shoulder in 1/3 to 1/2 of the patients due to irritation under the dome of diaphragm. Initially the patient may be shocked with a tachycardia but there is little change in the temperature. Respiration is shallow and the abdomen does not move with respiration. Tenderness and muscle guarding are constantly present over the right side of the abdomen.

STAGE OF PERITONEAL REACTION

During the secondary stage, the irritant gastric juice is diluted by the peritoneal exudates. The patient feels comfortable due to the buffering action of the fluid secreted. Symptoms are reduced but signs are still present. Muscular rigidity continues to be present. This stage is marked by two other features; obliteration of liver dullness and presence of shifting dullness. Evidence of free air within the abdominal cavity may be seen on a plain upright radiograph of the abdomen and chest in nearly 70% of the cases.

STAGE OF DIFFUSE PERITONITIS

In the tertiary stage, with the establishment of bacterial peritonitis, the patient has gone a step further towards the grave. The pinched and anxious face, sunken eyes and hollow cheeks – so called facies hippocratica, with rising pulse rate which is low in volume and tension, persistent vomiting, board like rigidity of the abdomen, increasing the distension of the abdomen all are evident in the terminal stage.

At times the spillage of the luminal contents is more of seepage and if seepage becomes contained in a smaller area, the pain though intensive, is located near the site of perforation and muscular rigidity is limited in extent. In posterior perforation the inflammatory reaction is contained in the lesser sac and symptoms may be obscure.[13,14]

FORME FRUSTE

When the breech in the wall of the stomach or duodenum is small and spontaneously sealed off rapidly before contamination of the general peritoneal cavity pain is less intense. Guarding and rigidity is present only in the epigastrium and right hypochondrium. Other areas of the abdomen are soft; this condition is known as forme fruste of acute free perforation.

CONFINED PEFORATION / GRADUAL PENETRATION

Penetration occurs when a peptic ulcer burrows through the wall of the stomach or duodenum but, instead of perforating freely into the peritoneal cavity, the crater bores into an adjacent organ[15]. Duodenal ulcers that involve the posterior wall of the bulb can penetrate into the pancreas. Penetrating gastric ulcers often involve the left lobe of the liver. Rarely, penetrating peptic ulcers can result in the development of fistulas between the duodenum and the common bile duct (choledochoduodenal fistula) or between the stomach and the colon (gastrocolic fistula).

Penetration can be associated with a change in the typical pattern of ulcer symptoms, patients may complain of an increasing intensity or longer duration of pain, or they may notice that the pain radiates into the back or that eating no longer relieves the discomfort.[16]

PERFORATION AND HAEMORRHAGE

The combination of perforation and hemorrhage occurs in either way.

1. Perforation occurring in the course of medical management of hemorrhage.
2. Onset of hemorrhage after a recent perforation.

INVESTIGATIONS

1. IMAGING STUDIES

a) X rays

i) Erect radiographs of the chest and a plain upright radiograph of the abdomen are the most common first line of diagnostic imaging when a perforated peptic ulcer is considered.

As little as 1 ml of free air may be visualized. Free air is present in 6% to 80% of cases.[17,18] In the upright view, curvilinear lucencies separate the most superior portion of the diaphragm from the liver on the right side and from the stomach and spleen on the left. An air-fluid level in the stomach should not be mistaken for free air. Usually the lateral margin of the air-fluid level can be seen extending to the lateral wall of the stomach, demarcated by serosal fat.

Causes of pseudopneumoperitoneum in a plain X-ray Abdomen are

- Chiliaditi syndrome
- Sub diaphragmatic fat
- Curvilinear pulmonary collapse
- Omental fat
- Subphrenic abscess with gas forming organisms
- Subpulmonary pneumothroax
- Intramural gas in pneumatosis intestinalis

ii) On the lateral decubitus view, the free air is usually best seen adjacent to the lateral margin of the liver, but in some patients the iliac portions of the peritoneum are more superior in location and free gas accumulates preferentially over the upper iliac bone.

iii) The supine view may occasionally be the only view ordered and available, especially if pneumoperitoneum is not suspected. Pneumoperitoneum can be detected in a supine view if free gas surrounds a gas-filed bowel loop. In this situation, the inner and outer margins of bowel wall are clearly seen (the Rigler sign). Some fat may normally outline the serosal surface of bowel loops, but in the presence of pneumoperitoneum the outer surface of the bowel is sharply marginated and more distinct than fat-outlined bowel. Small amounts of air rise to the most superior portions of the abdomen and may be seen outlining the anterior margin of the liver, forming an oblique or triangular lucency superimposed over the lower portion of the liver. A linear lucency overlying the medial mid-liver may represent free air in the fissure for the ligamentum teres. If large amounts of free air are present, air may outline the falciform ligament anterior to the liver, producing the “foot ball” sign, a large oval collection of air with a central soft tissue stripe produced by the falciform ligament outlined by surrounding gas. Air under the inferior abdominal wall may outline the umbilical folds the inverted – V sign. The Rigler sign and air collection overlying the liver are the mot common signs of free air on a supine abdominal view.[19]

B) Contrast Radiography

i) Contrast radiography using water-soluble diatrizoate meglumine [Gastrograffin] is useful in doubtful cases. In free perforation there is leakage of contrast into the peritoneal cavity.

ii)Gastrograffin administered contrast is also useful in diagnosis of sealed perforation to plan a conservative management as in the case of forme fruste.

C) Ultra Sonograms of the Abdomen

Localized gas collection related to bowel perforation may be detectable, particularly if it is associated with other sonographic abnormalities (e.g. thickened bowel loop).

The site of bowel perforation can be detected by sonography (e.g. gastric vs. duodenal perforation).

Ultra sonograms of the abdomen can also provide rapid evaluation of the liver, spleen, pancreas, kidneys, ovaries, adrenals and uterus, to rule out associated pathology.

D) CT Scans of the Abdomen

This modality can be a valuable investigative tool, providing differential morphologic information not obtainable with plain radiography or ultrasonography.

CT Scans may provide evidence of localized perforation (e.g., perforated duodenal ulcer) with leakage in the area of the gallbladder and right flank with or without free air being apparent.

2. LAB STUDIES

Complete Hemogram :

Parameters suggestive of infection (e.g., leukocytosis); Leukocytosis may be absent in elderly patients.

- Elevated packed blood cell volume suggests a shift of intravascular fluid.
- Blood culture for aerobic and anaerobic organisms.
- Liver function and renal function: Findings may be within reference ranges, when no preexisting disorder is present.

3. OTHER TESTS

Laparoscopy improves surgical decision making in patients with acute abdominal pain, particularly when the need for operation is uncertain.

DIFFERENTIAL DIAGNOSIS

- Acute appendicitis
- Cholecystitits, biliary colic
- Acute pancreatitits
- Typhoid fever
- Meckel's diverticulum
- Diverticular disease
- Ischemic colitis
- Inflammatory bowel disease
- Colitis

- Acute salphingitis
- Endometriosis
- Pelvic inflammatory disease
- Ovarian torsion
- Constipation

The non-abdominal conditions resembling perforation are

- Myocardial infarction
- Pleurisy
- Spontaneous pneumothroax
- Diabetes mellitus
- Acute porphyria

MANAGEMENT

Divided into conservative and operative management.

CONSERVATIVE MANAGEMENT

There are several studies advocating non-operative management in selected patients with a successful outcome.[20-21] The candidates who are tolerating the insult well and in whom perforation seems to have sealed can be managed conservatively. Resuscitation with intravenous fluids naso gastric suction and intravenous antibiotics and H_2 blockers resulted in mortality and mobility similar to those of operative management, but hospitalization is prolonged and incidence of subphrenic abscess is high. If non operative treatment is chosen then the patient will require frequent clinical evaluation, so that operative therapy can be initiated at the first sign of clinical deterioration.

OPERATIVE MANAGEMENT

The goals of operative management are as follows:

- To correct the anatomic problem
- To correct the cause of peritonitis
- To remove any foreign material in the peritoneal cavity that might inhibit WBC function and promote bacterial growth (food, bile, gastric and intestinal secretions).

PRE-OPERATIVE DETAILS

The initial priorities are resuscitation and analgesia.

- Correction of fluid and electrolyte imbalance: Extracellular fluid losses are replaced by colloids or crystalloids that have an electrolyte composition similar to plasma.
- Monitoring of Central venous pressure (CVP) in critically ill and / or elderly patients, in whom cardiac impairment may be exacerbated by large fluid loss.
- Administration of systemic antibiotics and establishing the likely organisms.
- Nasogastric suction to empty the stomach and reduce the risk of further vomiting.
- Urinary catheterization to assess urinary flow and adequacy of fluid replacement.
- Analgesics, such as morphine, in small intravenous doses, preferably as a continuous infusion.

OPERATIVE MANAGEMENT

1. Simple closure with dead omental patch.
2. Simple closure with live omental patch.
3. Simple closure with definitive procedure for ulcer.
4. Endoscopic closure of perforated ulcer.
5. Laparoscopic closure of perforated ulcer.
6. Closure with serosal patch

   These are the various target oriented operative techniques. All these techniques should be supplemented with thorough peritoneal lavage. Laparoscopic approach holds good in peritoneal lavage permitting irrigation of all corners of the peritoneal cavity.
7. Flank drain and conservative management is a non target oriented technique in patients of poor general conditions.

DEFINITIVE PROCEDURES FOR DUODENAL ULCER PERFORATION

1. Truncal vagotomy with suitable drainage procedure.
2. Highly selective vagotomy.
3. Taylor procedure (anterior seromyotomy with posterior truncal vagotomy).
4. Laparoscopic perforation closure, using intracorporeal suturing in a manner identical to open surgery, depending on the experience of surgeon, proximal gastric vagotomy or Taylor's procedure may be performed.

DEFINITIVE PROCEDURES FOR GASTRIC ULCER PERFORATION

1. Resection of ulcer and closure
2. Primary gastric resection with Billroth I anastomosis.

INDICATIONS FOR DEFINITIVE ULCER SURGERY

- Hemodynamically stable young patients
- Perforations for less than 24 hours
- No obvious co-morbidity
- Patients with long history of peptic ulcer
- Perforation of an ulcer during antisecretory agent
- Previous ulcer complications.

CONTRAINDICATIONS FOR DEFINITIVE ULCER SURGERY

- Associated medical conditions
- Delay in presentation of more than 24 hours
- Gross abdominal contamination with food.

POST OPERATIVE MANAGEMENT

- Intravenous replacement therapy: The aim of intravenous replacement therapy is to maintain intravascular volume and adequate hydration of the patient that can be monitored by CVP measurement and urinary output.
- Nasogastric drainage: Nasogastric drainage is continued until drainage becomes minimal. At this stage, the nasogastric tube may be removed.
- Antibiotics: the antibiotics commenced preoperatively are continued unless the results of cultures taken at the time of the operation reveal that the causative organisms are resistant to them.
- The goal of antibiotic therapy is to achieve levels of antibiotics at the site of infection that exceed the minimum inhibitory concentrations for the pathogens present.

- In the presence of intra-abdominal infections, gastrointestinal function is often impaired; therefore, oral antibiotics are not efficacious, and intravenous antibiotics are preferred.
- If no obvious improvement in the patient's condition occurs within 2-3 days, the following possibilities are considered.
    i. The initial operative procedure was inadequate.
    ii. Complications have occurred.
    iii. A super infection has occurred at a new site.
    iv. The dose of antibiotic is inadequate.
    v. The antibiotics used do not provide adequate coverage for anaerobes and gram-negative organisms.
- H_2 receptor antagonists or proton-pump inhibitors for a period of 6 – 8 weeks and
- A full regime of H. pylori eradication therapy to be started at the end of 8 weeks.

COMPLICATIONS

EARLY COMPLICATIONS

- Renal failure and fluid, electrolyte, and pH imbalance.
- Respiratory complications.
- Wound infection:
    i. Wound infection rates correlate with the bacterial load in the peritoneal fluid.
    ii. The judicious use of prophylactic antibiotics has been demonstrated to reduce the incidence of wound infection in contaminated and potentially contaminated wounds.

- Wound failure
- Wound failure (partial or total disruption of any or all layers of the operative wound) may occur early (i.e., wound dehiscence)
- The factors are associated with wound failure are malnutrition, sepsis, uremia, diabetes mellitus, corticosteroid therapy, obesity, heavy coughing, hematoma (with or without infection).
- Multiorgan failure and septic shock
    i. Septicemia is defined as proliferation of bacteria in the bloodstream resulting in systemic manifestations such as rigors, fever, hypothermia (in gram negative septicemia with endotoxemia), leukocytosis or leukopenia (in profound septicemia), tachycardia and circulatory collapse.
    ii. Septic shock is associated with loss of vasomotor tone, increased capillary permeability, myocardial depression, consumption of WBCs and platelets, dissemination of powerful vasoactive substances, such as histamine, serotonin, and prostaglandins, resulting in capillary permeability, complement activation and damage of capillary endothelium.
- Gram-negative infections are associated with a much worse prognosis than gram-positive infections, possibly because of associated endotoxemia.
- Localized abdominal abscess
- Entero cutaneous, fistula
- Deep vein thrombosis and pulmonary embolism.

LATE COMPLICATIONS

- Mechanical intestinal obstruction: Mechanical obstruction of the intestine is most often caused by postoperative adhesions.
- Incisional hernia

OUTCOME AND PROGNOSIS

Outcome is improved with early diagnosis and treatment. The factors increase the risk of death are advanced age, malnutrition, presence of preexisting underlying disease, presence of shock, delayed presentation, the nature and amount of contamination of the peritoneal fluid, perforation of greater size. Multiple regression analysis shows that the number of coexisting medical conditions, hypotension, and the duration of perforation are 3 independent risk factors contributing to a fatal outcome.[22] Multiple risk factors increase the mortality rate dramatically:[23]

No. of Risk factors	Mortality Rate
0	9%
1	10%
2	45.5%
3	100%

OTHER GASTRO DUODENAL PERFORATIONS

TRAUMATIC PERFORATION OF STOMACH

Penetrating injuries are more commonly associated with perforation of stomach than does blunt injury[24]. They may be caused due to stab injury with sharp knife or high velocity bullets. In most of these cases, extent of damage is

difficult to assess clinically due to alteration in the laminar layers of abdominal wall.

Nasogastric aspiration may be frank blood, X-ray showing gas under diaphragm is not contributory. Immediate surgical exploration is the management. Simple closure or gastric resection should be done.

TRAUMATIC PERFORATION OF DUODENUM

In blunt injury fixed pat of duodenum is affected more than the stomach, seat belt injuries are a well-recognized cause of duodenal rupture.

In Retroperitoneal rupture, pain in the epigastrium and back, with vomiting, epigastric and flank tenderness are present. X-ray shows presence of air in the region of right kidney or Psoas muscle may be outlined by air. Mobilization of duodenum by kocherization and closure of the rent should be done[24].

In Intraperitoneal rupture, there may be severe abdominal pain, X-ray shows air under diaphragm. Simple closure with proximal diversion is ideal.

IATROGENIC PERFORATION OF DUODENUM

Duodenal perforation is most common during ERCP with endoscopic sphincterotomy. This complication occurs in 0.3 to 2.1% of cases. Patients who have undergone Billroth II gastrectomy are at increased risk; duodenal perforation complicates 1.5 to 5% of the ERCPs in these patients. Perforations of the duodenum distal to its bulb, because of its retroperitoneal location, tend to be

locally contained and can present insidiously. Manifestations of contained duodenal perforation following ERCP can resemble those of ERCP – included pancreatitis, including hyperamylasemia[25]. Open transduodenal sphincteroplasty is complicated by duodenal perforation in 0.6% of cases[26].

Investigations include Plain Abdominal radiograph, contrast radiograph with Gastrograffin, CT scan.

Iatrogenic perforation incurred during endoscopy, if immediately recognized, can be repaired using endoscopic techniques. Intraperitoneal duodenal perforations require surgical repair.

PERFORATIONS OF SMALL BOWEL INCLUDING APPENDIX

The etiological factors for small intestinal perforations are:

INFECTIVE

Bacterial	:	Salmonella typhi, Mycobacterium tuberculosis, Clostridium perfringens, Campylobacter, Yersiniosis.
Fungal	:	Actinomyces
Viral	:	Cytomegalovirus
Parasitic	:	Ascariasis

Inflammatory :

Idiopathic, Inflammatory bowel disease, Necrotizing enterocolitis, Ischeamic enteirits, Radiation enteritis.

Traumatic	:	Blunt and penetrating injury
Diverticular disease	:	Meckel's, Jejunal and Ileal diverticulitis.
Malignancies	:	Lymphoma, Malignant melanoma.

Drug Induced.

TYPHOID ULCER PERFORATION

Typhoid fever remains endemic in tropical and subtropical countries, causative organism are S. typhi, paratyphi A and B, more common following the onset of monsoon, male to female ration is 3:1.

Typhoid fever is characterized by early septicemia phase with colonization of several organs such as liver, spleen, gall bladder, bones and small intestine.

Terminal ileum, in the region of Peyer's patches bears the brunt of infection with the formation of a longitudinal ulcerating lesions, on the antimesentric border, situated within 45 cm of ileocaecal valve. This ulceration may give rise to frank perforation at the end of third week of fever.

In the first week Peyer's patches becomes hyperemic and hyperplastic, necrosis in the second week. Ulceration in the third week, followed by healing or perforation in the fourth week.

The perforation is solitary in 85% of cases. Perforation of ulcerated Peyer's patch occurs in approximately 2% of cases[27].

Clinical features include fever, abdominal pain, tenderness, guarding, rigidity, and electrolyte imbalance. Plain X-ray may show air under diaphragm or multiple air fluid levels. A low WBC count before perforation, raises after perforation, Positive blood culture in the first week, positive widal test in the second week, positive stool culture in the third week are diagnostic.

MANAGEMENT

Resuscitation, correction of electrolyte imbalance and appropriate antibiotics.

The surgical procedures are:

- Single perforation with less contamination of abdominal cavity - Two-layer closure with peritoneal lavage.
- Multiple perforation with less contamination, ileal resection and end to end anastomosis.
- Perforation with heavy contamination of abdominal cavity requires exteriorization of the intestinal loops.
- Ileostomy also done for very sick, anemic, and high risk patients, with intensive monitoring for pH and electrolyte imbalance.

TUBERCULOSIS ULCER PERFORATION

Intestinal Tuberculosis is a rare cause of perforation. Commonest site is ileocaecal region[28]. Pathologically there are four types of intestinal tuberculosis – Hyperplastic, Ulcerative, Fibrotic, Ulcerofibrotic.

Perforation is usually solitary and occurs proximal to or at the site of stricture, diagnosis is aided by Mantoux, X-ray chest, peritoneal aspirate, and mesenteric node biopsy. Operative management includes wedge resection of the stenotic segment with perforation and end to end anastomosis under antituberculosis drug therapy.

MECKEL'S DIVERTICULUM

It is the remnant of vitellointestinal duct, present in 2% of population situated on the antimesentric border of small intestine, 2 feet from ileocaecal valve and usually 2 inches long. The presentations include – Severe hemorrhage, Intussusception, Meckel's diverticulitis, Chronic peptic ulceration, Intestinal obstruction.

Meckels diverticulitis can result in perforation. Management includes excision of the diverticulum with the wedge of adjacent ileum with an end to end anastomosis.

CROHN'S DISEASE

It is a chronic, transmural inflammatory bowel disease of gastrointestinal tract of unknown etiology. Crohn's disease can involve any part of the alimentary tract from the mouth to anus but most commonly affects small intestine and colon. Areas of diseased bowel separated by normal appearing bowel called 'skip areas' are pathagnomonic. Intestinal perforation with fistula formation or free perforation as a complication of Crohn's disease requires surgical intervention. Operative treatment of perforation should be limited to that segment

of the bowel involved with perforation and no attempt should be made to resect more bowel even though grossly evident disease may be apparent in the adjacent areas.

TUMORS OF SMALL BOWEL

Malignant tumors of small bowel include adenocarcinoma, malignant gastrointestinal stromal tumors and lymphomas.

Perforations are rare in cases of adenocarcinoma. Free perforation of malignant gastrointestinal occurs as a result of hemorrhagic necrosis in large masses.

Malignant lymphomas of the small bowel are most commonly found in the ileum, perforation may occur in up to 25% of patients.

The treatment includes resection of the tumor with a wide margin of normal bowel and a wedge of mesentry to remove immediate draining lymph nodes with primary anastomosis and repair of mesentry.

TRAUMATIC INJURIES OF SMALL BOWEL

Penetrating injuries are more common than blunt injuries. Traumatic ruptures involving mobile parts of small intestine are multiple. In blunt injuries the mechanisms involved are:

1. Crush injury between vertebrae and anterior abdominal wall.

2. Sudden increase in the intra abdominal pressure.
3. Tear at the junction of mobile and fixed portion of bowel due to deceleration.

Clinical features include features of peritoneal irritation and tenderness at the site of injury, diagnostic peritoneal lavage is of great value in detecting intra abdominal injuries, X-ray may reveal pneumoperitoneum. Operative management involves simple two layer closure if tear is small, resection needed when multiple tears within short segment.

IATROGENIC PERFORATIONS OF SMALL BOWEL

Incidents of small bowel perforation in laproscopy and Trocar suprapubic cystostomy are encountered rarely. ERCP can cause jejunal perforations in patients who have undergone Billroth II gastrectomy.

APPENDICULAR PERFORATIONS

Patients presenting late in the course of acute appendicitis may end up in Perforation. With the presence of luminal obstruction by a faecolith and acute inflammation, the appendix becomes edematous, ischemic and gangrenous. Without intervention, the gangrenous appendix will perforate with spillage of appendiceal contents into the peritoneal cavity. If this sequence occurs slowly, the inflammatory response and the omentum leading to localized peritonitis and an appendiceal abscess contain the appendix. If the process is not walled off, the patient may develop diffuse peritonitis.

Approximately 50% of cases of gangrenous or perforated appendicitis are associated with faecolith. At extremes of ages (below 5 years and above 60 years) the rate of perforation is in the region of 60%[29]. Immunocompromised individuals, diabetics, patients with pelvic appendix and previous abdominal surgeries are at higher risk for perforation.

The treatment includes appendectomy with peritoneal lavage.

COMPLICATIONS OF SMALL BOWEL PERFORATIONS

Wound infection, Intra abdominal abscess, Enterocutaneous / Faecal fistula, Portal pyemia, Adhesive obstruction, Reperforation.

PERFORATIONS OF LARGE BOWEL

AETIOLOGY

Infective

Bacterial : Paratyphoid B, Mycobacterium tuberculosis.

Fungal : Actinomyces

Parasitic : Entamoeba histolytica

Inflammatory

Ulcerative colitis

Diverticular Disease:

Volvulus

Traumatic : Blunt and Penetrating injuries.

Injury due to compressed air.

Iatrogenic : Rigid sigmoidoscopy and colonoscopy.

Malignancy :

AMOEBIC BOWEL PERFORATION

Entamoeba histolytica the causative organism has a worldwide distribution. The trophozoite form of the parasite is pathogenic. It makes its way into the follicles of Liberkuhn and submucus loculi are produced. Some of them burst through mucus membrane to become amoebic ulcers. The ulcers are bottlenecked because of undermined edges. 75% of the ulcers are confined to lower sigmoid and rectum. The trophozoites continue their activities in the base of the ulcer.

The most common sites of perforation are the caecum and rectosigmoid. Some times ulcer affects entire large bowels. The management includes segmental colectomy in localized disease and total colectomy with ileostomy and mucus fistula in generalized disease with administration of Metronidazole.

PERFORATION IN ULCERATIVE COLITIS

Ulcerative Colitis is a nonspecific inflammatory disease of the mucosa and submucosa of colon and rectum. Perforation in ulcerative colitis is a grave complication. It carries 50% mortality and accounts for 30% of all deaths due to ulcerative colitis. The risk appears to be highest during the severe attack of the disease with extensive inflammation resulting in Toxic megacolon. As perforation ensues the patients' condition suddenly deteriorates and the patient goes in to a state of shock due to faecal peritonitis. The condition is diagnosed by X-ray abdomen. Total colectomy with Hartmann's closure of the rectum or mucus fistula is the treatment of choice.

PERFORATION IN DIVERTICULAR DISEASE

Diverticula are herniations of the mucosa through the muscular layers of colon at the point of entry of segmental blood vessels from serosa to submucosal layer. Diverticular diseases are common in Western society with a prevalence of 30% in over 60 years of age, but the disease is asymptomatic in about 90% of cases. Diverticulitis is sometimes complicated by perforation. In acute perforation, peritonitis soon becomes general, may be purulent, and has a mortality of about 15%. Gross faecal peritonitis causes more than 50% mortality[30]. The surgical procedures are:

1. Primary resection and Hartmann's procedure
2. Primary resection and anastomosis after on table lavage in selected cases.
3. Suture of the perforation with drainage with or without proximal diversion.

PERFORATION IN MALIGNANCIES

In carcinoma of large intestine, perforation can be due to annular growth causing obstruction and tension gangrene and perforation of caecum due to malignant ulcer. There is a closed loop phenomenon between distal obstruction and proximal ileocaecal value. The intraluminal pressure raises and the brunt of the disease born by the caecum. This results in gangrene and perforation. Commonly perforation is localized with abscess formation or a fistulous communication between adjacent organs. The surgical resection depends on the site of the growth with exteriorization of the both ends.

TRAUMATIC PERFORATION

Traumatic Perforations of colon and rectum are due to penetrating injuries. The force required to damage the colon is considerable and so it is refractory to blunt injury. Blunt trauma accounts for 5% of colonic injuries. Rectal injuries occur in association with pelvic fractures. In intraperiotneal colonic injury present with signs of peritonitis, Diagnostic peritoneal lavage is helpful in diagnosis. In extrapertioneal colonic injury and in rectal injury diagnosis is difficult. Rectal injury should be considered in all patients with penetrating injury to the perineum and accidental high pressure air introduced from below. Management includes early resuscitation prophylactic antibiotics with the surgical options of (a) Primary closure of low risk colonic injuries, (b) Primary closure with proximal colostomy and (c) Resection and proximal colostomy.

COMPLICATIONS OF COLONIC PERFORATION

Faecal peritonitis, Abscess formation, Enterocutaneous fistula, Urinary or rectal fistula and Anastomotic leak.

MORBIDITY AND MORTALITY PREDICTORS

The factors influencing mortality and morbidity are elderly age group, delay by more than 24 hours, preoperative haemodynamic shock, coexisting medical illness, long-standing perforation, amount of peritoneal contamination and delay between onset of symptoms and surgery.

PATIENTS AND METHODS

This study was conducted in the Department of General Surgery, Government Rajaji Hospital, Madurai Medical College, Madurai, Tamilnadu for a period of 24 months from July 2004 to July 2006.

One hundred and thirty three cases of Gastro intestinal perforations were studied during the period.

The diagnosis was established by the Emergency Surgeon provisionally, based on the clinical presentation and supporting radiological evidence, in the ward, and definitive diagnosis established at the time of operation.

Based on the time interval between the hospital admission and surgery, the surgery was categorized into,

a) Immediate - Less than 4 hours

b) Same day - 4 to 24 hours

c) Delayed - more than 24 hours

Operative details included the site of the perforation, size of the perforation, nature and quantity of peritoneal soiling, the gross appearance of the bowel bearing the perforation and the nature of operation performed. Tissue biopsies for histologic confirmation were taken in appropriate cases.

Mortality was defined as death following surgery.

Morbidity was defined in terms of duration of hospital stay and associated complications following surgery.

Following details were observed from the case sheets and clinical examination.

- Patients name, age, sex, inpatient number.
- Clinical features and abdominal findings

- Delay in hours between admission and surgery
- Operative findings
- Procedure done
- Post operative complications
- Duration of hospital stay

INCLUSION CRITERIA

- Cases of acute perforation due to peptic ulcer disease.
- Cases of perforation of small bowel due to diseases.
- Cases of Appendicular perforation.
- Perforation of caecum and colon.
- Cases of traumatic perforations - both blunt and penetrating types.

EXCLUSION CRITERIA

- Cases of Oesophageal perforation / rupture
- Cases of perforations of hepatobiliary system.
- Cases of Iatrogenic perforation during laparotomy, and gynecological procedures.
- Cases of delayed presentation with shock and septicemia whose general condition did not warrant any operative management even after all resuscitative measures.

DISCUSSION

One hundred and thirty three cases of Gastro intestinal perforations were studied. Majority of the cases of perforations were the complications of peptic ulcer disease. Anatomically perforations were more common in the duodenum.

Table 1 : Anatomical distribution of perforations

S.No.	Site	No. of Cases	Percentage
1.	Duodenum	79	59.5
2.	Appendicular	18	13.5
3.	Ileal	16	12
4.	Gastric	12	9
5.	Jejunal	4	3
6.	Meckel's Diverticulum	2	1.5
7.	Colon	2	1.5
	Total	**133**	**100**

DUODENAL ULCER PERFORATION

There were 79 cases of perforation as a complication of duodenal ulcer.

AGE INCIDENCE:

Table 2 : Age Distribution of Duodenal Perforation

S.No.	Age	No. of Cases	Percentage
1.	< 19	2	2.53
2.	22 – 29	12	15.18
3.	30 – 39	21	26.58
4.	40 – 49	11	13.92
5.	50 – 59	15	18.98
6.	> 60	18	22.78
	Total	**79**	**100**

Duodenal perforations were more common in the age group of 30 – 39 years. The youngest case was 17 years and eldest case was 80 years. Average was 44 years.

COMPARATIVE STUDY

Study	Age Group (Years)
Gupta PS, Taluk dar RN, Neupave HC, Kathmandu University Med. J. 2003	51 – 60
Plummer, JM, McForlane ME, Nuwnham West Indian Med. J.	Male : < 50 Female : > 50
Ersumo D. W, Meskel, Y, Kotisso B, Ethiop Med. J. 2005	32.5 (Avg)
Kassis .M.S. Al Wattor	30 – 50
Norfolk UK	Male : 67.7 Female : 76.6
This study	44

SEX DISTRIBUTION

Six female patients of duodenal perforation were included in the study with the Male : Female Ratio of 12:1

Table 3 : Sex Distribution of Duodenal Perforation

S. No.	Sex	No. of Cases
1.	Male	73
2.	Female	6
	Total	**79**

COMPARATIVE STUDY

Study	Male : Female
Ersumo D. W, Meskel, Y, Kotisso B, Ethiop Med. J. 2005	7.2 : 1
Plummer, JM, McForlane ME, Nuwnham West Indian Med. J.	9 : 1
Norfolk UK	53 : 47
Kassis .M.S. Al Wattor	14.5 : 1
This Study	12 : 1

SOCIO ECONOMIC STATUS

72 patients belonged to low socio economic group.

53 patients were from rural area.

COMPARATIVE STUDY

Study	Rural
Kassis .M.S. Al Wattor	58%
This Study	67%

51 cases were smokers and 42 cases gave history of alcohol consumption while 8 patients gave history of alcohol intake before the perforation.

65 cases gave history of symptoms of peptic ulcer disease and 2 patients gave history of drug intake.

COMPARATIVE STUDY

Study	Rural
George stain	75%
Ersumo D. W, Meskel, Y, Kotisso B, Ethiop Med. J. 2005	78%
This Study	82.28%

RADIOLOGICAL SIGNS

The plain X-ray abdomen of 63 patients showed gas under diaphragm.

Table 4 : Pneumoperitoneum in Duodenal Perforation

Gas under diaphragm	No. of Cases	Percentage
Positive	63	79.75
Negative	16	20.25

COMPARATIVE STUDY

Study	Pneumoperitoneum
Grassir, Romano S, Pinto A, Romano L Eur. J. Radiology. 2004	85.5%
Meiser, Meissnan	70%
This Study	79.75%

All the 79 cases underwent some form of operative management. 16 cases underwent immediate surgery, 61 cases on the same day and 2 cases underwent delayed surgery.

Table 5 : Time interval between Admission and Surgery

Time Interval in Hrs.	Surgery	No. of Cases	Percentage
0 – 4	Immediate	16	21.25
4 – 24	Same day	61	77.21
> 24	Delayed	2	2.54

4 cases had sealed perforations which were reinforced with omental patch. In 5 cases whose general condition did not warrant anesthesia bilateral flank drainage was done. For the other 70 cases, simple closure of the perforation using 2 0 vicryl with live omental patch (Graham's patch) was done.

The peritoneal soiling was less than 500 ml in 8 cases.

Table 6 : Quantity of Peritoneal Soiling

Quantity of Peritoneal Soiling	No. of Cases	Percentage
< 500	8	10.81
500 – 2000	45	60.81
> 2000	21	28.38

The size of the perforation was ≥ 1 cm in 13 cases.

COMPARATIVE STUDY

Study	Size > 1 cm
Gupta PS, Taluk dar RN, Neupave HC, Kathmandu University Med. J. 2003	25%
This study	16.45%

No patient had previous or multiple perforations. In all cases, peritoneal toileting was done.

Table 7 : Comparison of Perforation size and Peritoneal Soiling

Average size of the Perforation	Average Quantity of Soiling
< 5 mm	970
5 – 10 mm	1600
> 10 mm	2400

One case with history of left sided inguinal hernia presented with clinical features suggestive of obstruction. X-ray of the patient did not reveal pneumoperitoneum. On Inguinal exploration of the obstructed hernia, the sac contained 250ml of pus with occlusion of its neck. Later, on laparotomy there was a 0.5 cm perforation in the first part of duodenum. The obstruction might have been precipitated by the muscular rigidity following perforation.

All the cases were advised to continue H_2 blockers or proton pump inhibitors and a course of H.pylori eradication therapy.

12 patients developed post operative complications - wound infection (8 cases), right basal pneumonitis (2 cases), burst abdomen (1 case) and entero-cutaneous fistula (1 case), the fistula closed spontaneously with conservative management.

Table 8 : Post-operative complications in duodenal perforation

Complications	No. of Cases	Percentage
Wound Infection	8	66.66
Basal pneumonitis	2	16.66
Burst Abdomen	1	8.33
Entero-cutaneous fistula	1	8.33

8 cases expired, 5 of which were the cases treated by flank drainage alone.

GASTRIC PERFORATIONS

Gastric perforations were found in 12 cases of the entire study group.

Table 9 : Age incidence of Gastric perforations

S.No.	Age	No. of Cases	Percentage
1.	22 – 29	1	8.33
2.	30 – 39	1	8.33
3.	40 – 49	3	25.00
4.	50 – 59	5	41.66
5.	> 60	2	16.66
	Total	**12**	**100**

10 male cases and 2 female cases with a male : female ratio of 5:1 were studied.

Table 10 : Sex Distribution in Gastric Perforation

S. No.	Sex	No. of Cases
1.	Male	10
2.	Female	2
	Total	**12**

Gastric perforation occurred more often in the sixth decade of life.

6 cases were smokers and 5 cases were alcoholic.

1 case operated for bilateral inguinal hernia developed gastric perforation in the post operative ward on the fifth post operative day. 1 case was encountered in the intensive cardiac care unit admitted for myocardial infarction.

Table 11 : Pneumoperitoneum in Gastric Perforation

Gas under diaphragm	No. of Cases	Percentage
Positive	9	75
Negative	3	25

Plain upright X-ray of the abdomen showed air under the diaphragm in 9 cases.

COMPARATIVE STUDY

Study	Pneumoperitoneum
Grassir, Romano S, Pinto A, Romano L Eur. J. Radiology. 2004	85.5%
Rodrignez – Sanjuan J.C.	96%
This Study	75%

Stab injury was the cause of lacerated injury over the anterior wall of stomach in one patient. The edges were trimmed and closed in 2 layers using 2.0 vicryl and 3.0 silk.

The other patients were treated by simple closure with omental patch after the edges were trimmed for histological study. One patient proved positive for malignancy.

5 patients developed post operative complication, 4 cases of wound infection and 1 case of entro-cutaneous fistula. 2 patients died of cardiac arrest.

ILEAL PERFORATION

16 cases of ileal perforations were included in the study.

AGE INCIDENCE :

Table 12 : Age incidence in Ileal Perforation

S.No.	Age	No. of Cases	Percentage
1.	< 19	2	12.5
2.	22 – 29	1	6.25
3.	30 – 39	3	18.75
4.	40 – 49	5	31.25
5.	50 – 59	3	18.75
6.	> 60	2	12.5
	Total	**16**	**100**

More common age group is 40-49 years. 12 male and 4 female cases were studied with a male:female ratio of 3:1.

Table 13 : Sex Distribution in Ileal Perforation

S. No.	Sex	No. of Cases
1.	Male	12
2.	Female	4
	Total	**16**

8 patients had history of fever for 2 weeks and 7 patients were diagnosed as typhoid positive by widal test.

COMPARATIVE STUDY OF TYPHOID PERFORATION

Study	Age Avg. in Years	Male : Female
Adesunkanmi AR, Ajao O G J R Coll Surg Edind. 1997	19.5	4 : 1
Salih Hosoglu, Mustafa Aldemin Am.Jou. of Epidemiology 2004	28.2	4 : 1
Waques Alam Jan M Israr JPMI	28.25	7 : 3
This Study	36.14	4 : 3

Among 4 cases of traumatic perforations, (3 blunt injury and 1 stab injury). 1 patient had associated spleenic injury and 1 patient had mesentric tear who were treated by spleenctomy and resection anastomosis respectively. In the non-traumatic group, resection anastomosis as done in 3 cases, 1 case with multiple ileal perforations and 1 case with mesentric lymph nodes and later confirmed of tuberculosis by histology.

Table 14 : Distribution of Illeal Perforation

Disease	No. of Cases	Percentage
Typhoid	7	43.75
Tuberculosis	1	6.25
Trauma	4	25
Others	4	25

12 cases underwent perforation closure in two layers using 2.0 vicryl and 3.0 silk. Peritoneal toileting was done for all cases.

4 patients developed post operative wound infection. 2 cases expired due to septicemia.

APPENDICULAR PERFORATION

A total of 18 cases of Appendicular perforation were in the study group.

AGE INCIDENCE :

Table 15 : Age incidence in Appendicular Perforation

S.No.	Age	No. of Cases	Percentage
1.	< 19	3	16.66
2.	22 – 29	3	16.66
3.	30 – 39	6	33.33
4.	40 – 49	3	16.66
5.	50 – 59	3	16.66
	Total	**18**	**100**

More common in the 4th decade.

Male : Female ratio of 11:7

Table 16 : Sex Distribution of Appendicular Perforation

S. No.	Sex	No. of Cases
1.	Male	11
2.	Female	7
	Total	**18**

15 patients had symptoms lasting for more than 48 hours before hospitalization.

7 patients had faecolith obstruction.

All patients were treated by appendectomy with peritoneal toileting.

3 cases developed wound infection post operatively.

JEJUNAL PERFORATIONS

A total of 4 cases were included in the study. 2 cases of traumatic perforation (Blunt injury) and 2 cases of non-traumatic perforation.

1 case was a sequlae of post gastro-jejunostomy and 1 case associated with a jejunal diverticulum.

3 cases were treated by perforation closure in two layers using 2.0 vicryl and 3.0 silk and the 1 case underwent resection anastomosis.

Post operatively 1 case developed wound infection.

COLONIC PERFORATION

2 cases of colonic perforations were encountered in the study period. 1 case of Bull gore injury with a tear of 0.5 cm in the descending colon with no peritoneal contamination which was closed primarily.

The other case had three perforations in the rectosigmoid of amoebic etiology, treated by proximal colostomy. Patient had post operative wound infection.

PERFORATION OF MECKEL'S DIVERTICULUM

2 cases of perforated Meckel's diverticulitis were treated by resection anastomosis. No post operative complications.

OPERATIVE PROCEDURE – MORBIDITY AND MORTALITY

Site	Operative Procedure	No. of Cases	Morbidity	Mortality
Duodenum	SC with OP	74	12	3
	B/L flank drain	5	--	5
Stomach	SC with OP	11	4	2
	Closure in 2 layers	1	1	--
Ileum	Closure in 2 layers	12	2	1
	Resection & anastomosis	4	2	1
Jejunum	Closure in 2 layers	3	1	--
	Resection & anastomosis	1	--	--
Appendix	Appendectomy	18	3	--
Colon	Primary Closure	1	--	--
	Colostomy	1	1	--
Meckel's Diverticulum	Resection & anastomosis	2	--	--

MORTALITY

12 cases in the study expired due to the following reasons.

Age	Sex	Pathology	Procedure	Cause of death
60	M	DU – Perforation	B/L Flank Drain	Septicemic Shock
59	M	DU – Perforation	B/L Flank Drain	Septicemic Shock
40	M	DU – Perforation	B/L Flank Drain	Cardiac Arrest
61	M	DU – Perforation	B/L Flank Drain	Septicemic Shock
60	M	DU – Perforation	B/L Flank Drain	Septicemic Shock
73	M	DU – Perforation	SC with OP	Cardiac Arrest
65	M	DU – Perforation	SC with OP	Cardiac Arrest
80	M	DU – Perforation	SC with OP	Septicemic Shock
50	M	Gastric Perforation	SC with OP	Cardiac Arrest
50	F	Gastric Perforation	SC with OP	Cardiac Arrest
70	M	Ileal Perforation	Closure in 2 layers	Septicemic Shock
40	M	Ileal Perforation	Resection & anastomosis	Septicemic Shock

8 cases died of Septicemia and 4 cases due to cardiac arrest. Among the 12 cases 1 was female. The deaths occurred usually on the 2nd or 3rd post-operative day.

CONCLUSION

- Duodenal ulcer perforation was the commonest cause of gastrointestinal perforation with a male preponderance.
- More common in the fourth decade of life.
- More common in the lower socio-economic class of people.
- Smoking and alcohol were aggravating factors.
- Perforation was the first manifestation of peptic ulcer disease in a small percentage of patients.
- The role of nonsteroidal anti-inflammatory drugs as the cause of perforation was little in this study group.
- Radiological evidence of pneumoperitoneum could not be established in nearly one third of the patients.
- Simple closure with omental patch with thorough peritoneal toileting was very much effective.
- Definitive ulcer surgery was not warranted in the emergency and treatment with H_2 blockers and H. pylori eradication achieved good control over the disease in the follow up period.
- The prognostic indicators were early hospitalization, adequate fluid replacement and no co-existing medical illness.
- Gastric perforations were common in the sixth decade.
- The role of biopsy in gastric perforation was established with a case proving positive for malignancy.

- Appendicular perforation was the second common gastrointestinal perforation in the study.
- Delayed hospitalization was the major cause of perforation in appendicitis.
- Ileal perforations were mostly due to typhoid ulcer perforations.
- Jejunal perforations were rare and trauma was the single major cause of jejunal perforation.
- Closure in two layers was very much effective in small bowel perforations.
- Inspite of recent advances in closing duodenal perforation by laparoscopy and by other means, still simple closure with omental patch was widely practiced in the study group.
- The most common post-operative complication was wound infection.
- Deaths were due to septicemia and cardiac arrest.
- The actual mortality was higher than the mortality in the study group since cases of delayed presentation with shock and septicemia did not warrant anesthesia and were excluded from the study group.

PROFORMA

GASTRO INTESTINAL PERFORATIONS

CASE NO	:	IP NO	:
NAME	:	AGE & SEX	:
OCCUPATION	:		
ADDRESS	:		
DATE OF ADMISSION	:	DATE OF SURGERY:	
DATE OF DISCHARGE	:		

PRESENTING COMPLAINTS

1. PAIN ABDOMEN :
2. ABDOMINAL DISTENSION :
3. CONSTIPATION :
4. OBSTIPATION :
5. VOMITING :
6. OTHERS :

HISTORY OF PRESENT ILLNESS

I. PAIN ABDOMEN

a) MODE OF ONSET :

b) DURATION :

c) SITE :

d) RADIATION :

e) NATURE :

f) AGGRAVATING / RELIEVING FACTOR :

II. ABDOMINAL DISTENSION

a) DURATION :

b) DIFFUSE / LOCALISED :

c) IF LOCALISED SITE :

III. VOMITING

a) DURATION :

b) FREQUENCY :

c) CHARACTER :

d) VOMITUS :

e) RELATION TO PAIN :

IV. CONSTIPATION / OBSTIPATION :

V. OLIGURIA :

VI. OTHERS :

PAST HISTORY

1. ULCER PAIN :
2. HAEMETEMESIS :
3. MELAENA :
4. PREVIOUS SURGERY :

PERSONAL HISTORY

1. SMOKER :
2. ALCOHOLIC :
3. DIET :

MENSTURAL HISTORY

LAST MENSTURAL PERIOD :

EXAMINATION

I. GENERAL EXAMINATION

CONSCIOUSNESS	:	ATTITUDE	:
HYDRATION	:	APPERANCE	:
PULSE	:	BLOOD PRESSURE	:
RESPIRATORY RATE	:	TEMPERATURE	:
ANEMIA	:	JAUNDICE	:

II. EXAMINATION OF THE ABDOMEN

INSPECTION

a) SHAPE :

b) SKIN :

c) SCAR :

d) SWELLING :

e) UMBLICUS :

f) RESPIRATORY MOVEMENTS :

g) PERISTALTIC MOVEMENTS :

h) HERNIAL SITES :

PALPATION

a) TEMPERATURE :

b) TENDERNESS :

c) GUARDING :

d) RIGIDITY :

e) MASS :

f) LIVER / SPLEEN :

PERCUSSION

a) LIVER DULLNESS :

b) SHIFTING DULLNESS :

AUSCULTATION

BOWEL SOUNDS :

EXTERNAL GENITALIA :

DIGITAL RECTAL EXAMINATION :

III. SYSTEMIC EXAMINATION

a) CARDIOVASCULAR SYSTEM :

b) RESPIRATORY SYSTEM :

c) CENTRAL NERVOUS SYSTEM :

INVESTIGATIONS

1) BLOOD GROUPING :

2) HAEMOGLOBIN :

3) BLOOD SUGAR :

4) BLOOD UREA :

5) SERUM CREATININE :

6) PLAIN X-RAY ABDOMEN ERECT :

7) ULTRASOUND ABDOMEN :

CLINICAL DIAGNOSIS:

MANAGEMENT

CONSERVATIVE / OPERATIVE :

PREOPERATIVE RESUSCITATION :

IV FLUID / BLOOD TRANSFUSION :

OPERATIVE NOTES

DATE :

ANESTHESIA :

PEROPERATIVE FINDINGS :

FINAL DIAGNOSIS

POSTOPERATIVE COMPLICATIONS

FOLLOW UP

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ANNEXURE - PROFORMA

GASTRO INTESTINAL PERFORATIONS

CASE NO	**:**	**IP NO**	**:**
NAME	**:**	**AGE & SEX**	**:**
OCCUPATION	**:**		
ADDRESS	**:**		
DATE OF ADMISSION	**:**	**DATE OF SURGERY:**	
DATE OF DISCHARGE	**:**		

PRESENTING COMPLAINTS

1. **PAIN ABDOMEN :**
2. **ABDOMINAL DISTENSION :**
3. **CONSTIPATION :**
4. **OBSTIPATION :**
5. **VOMITING :**
6. **OTHERS :**

HISTORY OF PRESENT ILLNESS

I. PAIN ABDOMEN

a) **MODE OF ONSET :**

b) **DURATION :**

c) **SITE :**

d) **RADIATION :**

e) **NATURE :**

f) **AGGRAVATING / RELIEVING FACTOR :**

II. ABDOMINAL DISTENSION

a) **DURATION :**

b) **DIFFUSE / LOCALISED :**

c) **IF LOCALISED SITE :**

III. VOMITING

a) **DURATION :**

b) **FREQUENCY :**

c) **CHARACTER :**

d) **VOMITUS :**

e) **RELATION TO PAIN :**

IV. CONSTIPATION / OBSTIPATION :

V. OLIGURIA :

VI. OTHERS :

PAST HISTORY

1. **ULCER PAIN :**
2. **HAEMETEMESIS :**
3. **MELAENA :**
4. **PREVIOUS SURGERY :**

PERSONAL HISTORY

1. **SMOKER :**
2. **ALCOHOLIC :**
3. **DIET :**

MENSTURAL HISTORY

LAST MENSTURAL PERIOD :

EXAMINATION

I. GENERAL EXAMINATION

CONSCIOUSNESS	**:**	**ATTITUDE**	**:**
HYDRATION	**:**	**APPERANCE**	**:**
PULSE	**:**	**BLOOD PRESSURE:**	
RESPIRATORY RATE	**:**	**TEMPERATURE**	**:**
ANEMIA	**:**	**JAUNDICE**	**:**

II. EXAMINATION OF THE ABDOMEN

INSPECTION

a) **SHAPE** :

b) **SKIN** :

c) **SCAR** :

d) **SWELLING** :

e) **UMBLICUS** :

f) RESPIRATORY MOVEMENTS :

g) PERISTALTIC MOVEMENTS :

h) HERNIAL SITES :

PALPATION

a) TEMPERATURE :

b) TENDERNESS :

c) GUARDING :

d) **RIGIDITY** :

e) **MASS** :

f) LIVER / SPLEEN :

PERCUSSION

a) **LIVER DULLNESS :**

b) **SHIFTING DULLNESS :**

AUSCULTATION

BOWEL SOUNDS :

EXTERNAL GENITALIA :

DIGITAL RECTAL EXAMINATION :

III. SYSTEMIC EXAMINATION

a) CARDIOVASCULAR SYSTEM :

b) RESPIRATORY SYSTEM :

c) CENTRAL NERVOUS SYSTEM :

INVESTIGATIONS

1) **BLOOD GROUPING :**

2) **HAEMOGLOBIN :**

3) **BLOOD SUGAR :**

4) **BLOOD UREA :**

5) **SERUM CREATININE :**

6) **PLAIN X-RAY ABDOMEN ERECT :**

7) **ULTRASOUND ABDOMEN :**

CLINICAL DIAGNOSIS:

MANAGEMENT

CONSERVATIVE / OPERATIVE :

PREOPERATIVE RESUSCITATION :

IV FLUID / BLOOD TRANSFUSION :

OPERATIVE NOTES

DATE :

ANESTHESIA :

PEROPERATIVE FINDINGS :

FINAL DIAGNOSIS

POSTOPERATIVE COMPLICATIONS

FOLLOW UP